```
FILE 'HOME' ENTERED AT 09:07:19 ON 16 MAY 2002

=> fil reg

=> s nordihydroguaiaretic acid/cn
L1           1 NORDIHYDROGUAIARETIC ACID/CN

=> d

L1   ANSWER 1 OF 1  REGISTRY  COPYRIGHT 2002 ACS
RN   500-38-9  REGISTRY
CN   1,2-Benzenediol, 4,4'-(2,3-dimethyl-1,4-butanediyl)bis-  (9CI)  (CA INDEX
     NAME)
OTHER CA INDEX NAMES:
CN   Pyrocatechol, 4,4'-(2,3-dimethyltetramethylene)di-  (8CI)
OTHER NAMES:
CN   β,γ-Dimethyl-α,δ-bis(3,4-dihydroxyphenyl)butane
CN   1,4-Bis(3,4-dihydroxyphenyl)-2,3-dimethylbutane
CN   4,4'-(2,3-Dimethyl-1,4-butanediyl)bis(pyrocatechol)
CN   4,4'-(2,3-Dimethyltetramethylene)dipyrocatechol
CN   Butane, 1,4-bis(3,4-dihydroxyphenyl)-2,3-dimethyl-
CN   Dihydronorguaiaretic acid
CN   Dinorguaiaretic acid, dihydro-
CN   NDGA
CN   Nordihydroguaiaretic acid
CN   Norguaiaretic acid, dihydro-
FS   3D CONCORD
DR   1413-68-9
MF   C18 H22 O4
CI   COM
LC   STN Files:   AGRICOLA, ANABSTR, BEILSTEIN*, BIOBUSINESS, BIOSIS,
       BIOTECHNO, CA, CABA, CANCERLIT, CAOLD, CAPLUS, CASREACT, CBNB, CEN,
       CHEMCATS, CHEMLIST, CIN, CSCHEM, DDFU, DIOGENES, DRUGU, EMBASE, HODOC*,
       IFICDB, IFIPAT, IFIUDB, IPA, MEDLINE, MRCK*, NAPRALERT, NIOSHTIC, PROMT,
       RTECS*, SPECINFO, TOXCENTER, USPATFULL, VETU
         (*File contains numerically searchable property data)
     Other Sources:   EINECS**
         (**Enter CHEMLIST File for up-to-date regulatory information)
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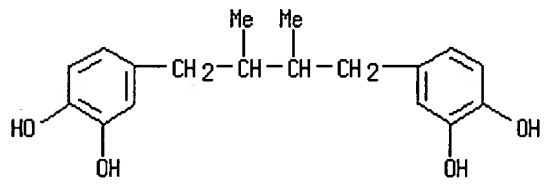


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**PROPERTY DATA AVAILABLE IN THE 'PROP' FORMAT**

            1093 REFERENCES IN FILE CA (1967 TO DATE)
              19 REFERENCES TO NON-SPECIFIC DERIVATIVES IN FILE CA
            1099 REFERENCES IN FILE CAPLUS (1967 TO DATE)
              17 REFERENCES IN FILE CAOLD (PRIOR TO 1967)


=> sel rn name l1
E1 THROUGH E11 ASSIGNED

=> fil medl capl biosis uspatful

=> s e1-11
   2 FILES SEARCHED...

SEARCH ENDED BY USER
L2        7006 (".BETA.,.GAMMA.-DIMETHYL-.ALPHA.,.DELTA.-BIS(3,4-DIHYDROXYPHENY
               L)BUTANE"/BI OR "BUTANE, 1,4-BIS(3,4-DIHYDROXYPHENYL)-2,3-DIMETH
               YL-"/BI OR "DIHYDRONORGUAIARETIC ACID"/BI OR "DINORGUAIARETIC
               ACID, DIHYDRO-"/BI OR NDGA/BI OR "NORDIHYDROGUAIARETIC ACID"/BI
```

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               OR "NORGUAIARETIC ACID, DIHYDRO-"/BI OR "1,4-BIS(3,4-DIHYDROXYPH
               ENYL)-2,3-DIMETHYLBUTANE"/BI OR "4,4'-(2,3-DIMETHYL-1,4-BUTANEDI
               YL)BIS(PYROCATECHOL)"/BI OR "4,4'-(2,3-DIMETHYLTETRAMETHYLENE)DI
               PYROCATECHOL"/BI OR 500-38-9/BI)

=> s obes? or antiobes? or weight gain or wight loss or overweigh?
L3      248201 OBES? OR ANTIOBES? OR WEIGHT GAIN OR WIGHT LOSS OR OVERWEIGH?

=> s l2 and l3

SEARCH ENDED BY USER
L4            6 L2 AND L3

=> s l2 and l3

s l2 and l3L5           24 L2 AND L3

=> s l2 and l3
L6           24 L2 AND L3  d his

=> d his

     (FILE 'HOME' ENTERED AT 09:07:19 ON 16 MAY 2002)

     FILE 'REGISTRY' ENTERED AT 09:07:42 ON 16 MAY 2002
L1             1 S NORDIHYDROGUAIARETIC ACID/CN
                 SEL RN NAME L1

     FILE 'MEDLINE, CAPLUS, BIOSIS, USPATFULL' ENTERED AT 09:08:46 ON 16 MAY
     2002
L2          7006 S E1-11
L3        248201 S OBES? OR ANTIOBES? OR WEIGHT GAIN OR WIGHT LOSS OR OVERWEIGH?
L4             6 S L2 AND L3
L5            24 S L2 AND L3
L6            24 S L2 AND L3

=> s l2 (S) l3

     (FILE 'HOME' ENTERED AT 09:07:19 ON 16 MAY 2002)

     FILE 'REGISTRY' ENTERED AT 09:07:42 ON 16 MAY 2002
L1             1 S NORDIHYDROGUAIARETIC ACID/CN
                 SEL RN NAME L1

     FILE 'MEDLINE, CAPLUS, BIOSIS, USPATFULL' ENTERED AT 09:08:46 ON 16 MAY
     2002
L2          7006 S E1-11
L3        248201 S OBES? OR ANTIOBES? OR WEIGHT GAIN OR WIGHT LOSS OR OVERWEIGH?
L4             6 S L2 AND L3
L5            24 S L2 AND L3
L6            24 S L2 AND L3

=> d his
d hisL7             1 L2 (S) L3

=> d

     (FILE 'HOME' ENTERED AT 09:07:19 ON 16 MAY 2002)

     FILE 'REGISTRY' ENTERED AT 09:07:42 ON 16 MAY 2002
L1             1 S NORDIHYDROGUAIARETIC ACID/CN
                 SEL RN NAME L1

     FILE 'MEDLINE, CAPLUS, BIOSIS, USPATFULL' ENTERED AT 09:08:46 ON 16 MAY
     2002
L2          7006 S E1-11
L3        248201 S OBES? OR ANTIOBES? OR WEIGHT GAIN OR WIGHT LOSS OR OVERWEIGH?
L4             6 S L2 AND L3
L5            24 S L2 AND L3
L6            24 S L2 AND L3
L7             1 S L2 (S) L3

=>

L7   ANSWER 1 OF 1  USPATFULL
```

Full Text
AN 1998:131760 USPATFULL
TI Use of bisphenolic compounds to treat type II diabetes
IN Khandwala, Atul S., San Carlos, CA, United States
Luo, Jian, Brisbane, CA, United States
PA Shaman Pharmaceuticals, Inc., South San Francisco, CA, United States
(U.S. corporation)
PI US 5827898 19981027
AI US 1996-726591 19961007 (8)
DT Utility
FS Granted
LN.CNT 824
INCL INCLM: 514/734.000
INCLS: 514/866.000
NCL NCLM: 514/734.000
NCLS: 514/866.000
IC [6]
ICM: A61K031-05
EXF 514/734; 514/866
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

=> d ibib abs kwic

L7 ANSWER 1 OF 1 USPATFULL
Full Text

ACCESSION NUMBER:	1998:131760 USPATFULL		
TITLE:	Use of bisphenolic compounds to treat type II diabetes		
INVENTOR(S):	Khandwala, Atul S., San Carlos, CA, United States Luo, Jian, Brisbane, CA, United States		
PATENT ASSIGNEE(S):	Shaman Pharmaceuticals, Inc., South San Francisco, CA, United States (U.S. corporation)		
	NUMBER	KIND	DATE
PATENT INFORMATION:	US 5827898		19981027
APPLICATION INFO.:	US 1996-726591		19961007 (8)
DOCUMENT TYPE:	Utility		
FILE SEGMENT:	Granted		
PRIMARY EXAMINER:	Jordan, Kimberly		
LEGAL REPRESENTATIVE:	Pennie & Edmonds LLP		
NUMBER OF CLAIMS:	3		
EXEMPLARY CLAIM:	1		
NUMBER OF DRAWINGS:	6 Drawing Figure(s); 6 Drawing Page(s)		
LINE COUNT:	824		

CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB This invention is directed to methods for treatment of non-insulin-dependent diabetes mellitus, for reducing blood glucose levels, or hyperglycemia. The methods entail administering to a mammal in need of such treatment a therapeutically effective amount of a composition whose active ingredient consists essentially of a compound of the following structure or a pharmaceutically acceptable salt thereof: ##STR1## In the compound R and R' are independently H or a C1 -C20 alkyl or C2 -C20 alkenyl group which may be substituted or unsubstituted. Alternatively, R and R' are such that together a cycloalkyl or cycloalkenyl ring is formed. In the chain linking the two phenolic derivatives each of (C(R).dbd.C(R')) or (C(R)(R')) are the same or different. A and A' are independently C2 -C20 acylamino, C2 -C20 acyloxy, C2 -C20 alkanoyl, C2 -C20 alkoxycarbonyl, C1 -C20 alkoxyl, C1 -C20 alkyl amino, C2 -C20 alkyl carboxyl, amino, C2 -C20 carbalkoxyl, carboxyl, cyano, halo, hydroxy. B and B' are independently H, C2 -C20 alkanoyl, C3 -C20 alkenoyl, C2 -C20 alkenyl, C2 -C20 alkoxycarbonyl, C1 -C20 alkyl, aroyl, aralkanoyl, C2 -C20 carbamoyl, or phosphate. The invention is also directed to methods of treatment using a bisphenolic compound in conjunction with another hypoglycemic or hypolipidemic agent.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.
DETD The following experiments demonstrate that the compounds described herein, for example **NDGA**, produce a significant and consistent hypoglycemic effect on **obese** diabetic mice, i.e., an art recognized model of diabetes mellitus. Further demonstrated are **NDGA**'s beneficial effects on glucose tolerance and the ability to stimulate glucose

transport in adipocytes.

DETD This experiment illustrates the effectiveness of **NDGA** in reducing plasma glucose levels in **obese** diabetic db/db mice, a model recognized by those skilled in the art as being a representative model of non-insulin-dependent diabetes. . .

DETD Genetically altered **obese** diabetic mice (designated C57BL/Ks-db/db) were purchased from The Jackson Laboratory (Bar Harbor, Me., USA), and served as experimental animals. Male. . . at the start of the study. Diabetic mice designated C57BLIKS-db/db received, orally by gavage, daily for 2 days either vehicle, **NDGA** administered at 150 mg/kg bid (9:00 and 17:00), 250 and 350 mg/kg q.d., or metformin [250 mg (1510 mol)/kg q.d].. . .

DETD This experiment illustrates the effectiveness of **NDGA** in reducing plasma glucose levels in **obese** diabetic ob/ob mice, a model recognized by those skilled in the art as being a representative model of non-insulin-dependent diabetes. . .

DETD FIG. 3 and 4 show the effect of **NDGA** in the oral glucose tolerance test. **NDGA** at all dosage-levels significantly suppressed the postprandial glucose levels at all timepoints after the glucose load. The oral glucose tolerance. . . was significantly improved as indicated by the area under the curve. This effect was dose dependent. These data indicate that **NDGA** enhances glucose utilization and improves the rate of glucose disposal in an animal model of insulin resistance, **obesity**, and NIDDM.

```
=> dup rem l6; focus
PROCESSING COMPLETED FOR L6
L8            24 DUP REM L6 (0 DUPLICATES REMOVED)


PROCESSING COMPLETED FOR L8
L9            24 FOCUS L8 1-

=> d ibib abs kwic 1-5

L9   ANSWER 1 OF 24  USPATFULL
Full Text
ACCESSION NUMBER:         1998:131760  USPATFULL
TITLE:                    Use of bisphenolic compounds to treat type II diabetes
INVENTOR(S):              Khandwala, Atul S., San Carlos, CA, United States
                          Luo, Jian, Brisbane, CA, United States
PATENT ASSIGNEE(S):       Shaman Pharmaceuticals, Inc., South San Francisco, CA,
                          United States (U.S. corporation)

                               NUMBER        KIND    DATE
                          --------------- ------ --------
PATENT INFORMATION:       US 5827898               19981027
APPLICATION INFO.:        US 1996-726591           19961007  (8)
DOCUMENT TYPE:            Utility
FILE SEGMENT:             Granted
PRIMARY EXAMINER:         Jordan, Kimberly
LEGAL REPRESENTATIVE:     Pennie & Edmonds LLP
NUMBER OF CLAIMS:         3
EXEMPLARY CLAIM:          1
NUMBER OF DRAWINGS:       6 Drawing Figure(s); 6 Drawing Page(s)
LINE COUNT:               824
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB     This invention is directed to methods for treatment of
       non-insulin-dependent diabetes mellitus, for reducing blood glucose
       levels, or hyperglycemia. The methods entail administering to a mammal
       in need of such treatment a therapeutically effective amount of a
       composition whose active ingredient consists essentially of a compound
       of the following structure or a pharmaceutically acceptable salt
       thereof: ##STR1## In the compound R and R' are independently H or a
       C1 -C20 alkyl or C2 -C20 alkenyl group which may be
       substituted or unsubstituted. Alternatively, R and R' are such that
       together a cycloalkyl or cycloalkenyl ring is formed. In the chain
       linking the two phenolic derivatives each of (C(R).dbd.C(R')) or
       (C(R)(R')) are the same or different. A and A' are independently C2
       -C20 acylamino, C2 -C20 acyloxy, C2 -C20
       alkanoyl, C2 -C20 alkoxycarbonyl, C1 -C20 alkoxyl,
       C1 -C20 alkyl amino, C2 -C20 alkyl carboxyl, amino,
       C2 -C20 carbalkoxyl, carboxyl, cyano, halo, hydroxy. B and B'
       are independently H, C2 -C20 alkanoyl, C3 -C20
```

alkenoyl, C2 -C20 alkenyl, C2 -C20 alkoxycarbonyl, C1 -C20 alkyl, aroyl, aralkanoyl, C2 -C20 carbamoyl, or phosphate. The invention is also directed to methods of treatment using a bisphenolic compound in conjunction with another hypoglycemic or hypolipidemic agent.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

SUMM 2.2 CHEMISTRY AND BIOLOGY OF **NDGA** AND OTHER BISPHENOLIC COMPOUNDS

SUMM 6. EXAMPLE: HYPOGLYCEMIC ACTIVITY OF **NDGA**

SUMM . . . treatment of hypoglycemia and hypoglycemia associated abnormalities in mammals. The method utilizes the bisphenolic compounds described herein including nordihydroguiaretic acid (**NDGA**), **NDGA** derivatives and **NDGA** analogs.

SUMM 2.2 CHEMISTRY AND BIOLOGY OF **NDGA** AND OTHER BISPHENOLIC COMPOUNDS

SUMM Nordihydroguiaretic Acid (**NDGA**) is also known as (R*, S*)-4,4'-(2,3-dimethyl-1,4-butanediol)bis[1,2-benzenediol]; meso-4,4'-(2,3'-dimethyletramethylene dipyrocatechol; 2,3-bis(3,4'-dihydroxybenzyl)butane; β,γ-dimethyl-α,δ-bis(3,4-dihydroxyphenylbutane); masoprocol; CHX-100; and ACTINEX®. The structure is shown below. ##STR2##

SUMM . . . Schroeter et al., 1918, Ber. 51:1587; R. D. Haworth et al., 1934, J. Chem. Soc., pp. 1423). The synthesis of **NDGA** was reported by Lieberman (Lieberman et al., 1947, J. Am. Chem. Soc., 69:1540) and the stereochemistry of the naturally occurring. . .

SUMM The use as of **NDGA** as an antioxidant was described by Lauer in U.S. Pat. No. 2,373,192. **NDGA** has been used as an antioxidant to inhibit rancidity of fats and as a stabilizer of pharmaceutical preparations.

SUMM Other workers have noted that **NDGA** has an antiproliferative effect on cultured keratinocytes and on cultured glioma cells (Wilkinson and Orenberg, 1987, Int. J. Dermatol., 26:660;. . . an inhibitor of the lipoxygenase pathway of arachidonic acid and useful for the treatment of psoriasis (U.S. Pat. No. 4,708,964). **NDGA** has been marketed for the topical treatment of actinic keratoses under the trademark ACTINEX® or the generic name masoprocol.

SUMM **NDGA** and certain analogs have also been reported to reduce cholesterol and triglyceride levels, specifically hyperlipidemia (U.S. Pat. No. 3,934,034) and. . . reported that 2,2'-alkylidene bisdialkyl phenols lower serum cholesterol. Vanadium and niobium complexes of a large variety of catechol derivatives including **NDGA** were claimed to be hypocholesterolemic, hypolipidemic and to be useful for the treatment of diabetes (PCT Publication No. WO 93/14751).. . .

SUMM **NDGA** is known as a lipoxygenase inhibitor. There is considerable confusion in the literature about the effects of arachidonic acid metabolites. . . and lipoxygenase inhibitors on insulin secretion (A. M. Band, et al., 1994, Pharmacology Communications, p. 285). Numerous publications report that **NDGA** and numerous other lipoxygenase inhibitors inhibit the release of glucose induced insulin secretion (see for example S. Yamamoto, et al.,. . .

SUMM Surprisingly it has now been discovered that the bisphenolic compounds, including nordihydroguiaretic acid, **NDGA**, its stereoisomers, analogs and derivatives as illustrated below, are effective in lowering blood sugar and in the treatment of diabetes,. . .

DRWD FIG. 1 is a histogram showing the plasma glucose levels of diabetic db/db mice treated with varying doses of **NDGA**. (*P<0.05, **P<0.01, **P<0.001 (Anova, one factor) N.dbd.8).**

DRWD . . . glucose levels of diabetic ob/ob mice treated either with a 250mg/kg dose of Metformin or with the same dose of NDGA. (*P<0.05, **P<0.01, ***P<0.001 (Anova)).

DRWD FIG. 3 shows the results of an oral glucose tolerance test for varying doses of **NDGA**. (***P<0.0001 (Anova, one factor)).

DRWD FIG. 4 shows the area under of the curve (AUC) for the oral glucose tolerance test of **NDGA**. (***P<0.001 (Anova, one factor)).

DRWD FIG. 5 shows the effect of **NDGA** on basal 2-deoxyglucose uptake in 3T3-L1 adipocytes.

DRWD FIG. 6 shows the effect of **NDGA** on insulin-stimulated 2-deoxyglucose uptake in 3T3-L1 adipocytes.

DETD . . . above, these compounds may be used alone or in combination with other known antidiabetic or hypolipidemic agents. The compounds including **NDGA**, derivatives and analogs, or pharmaceutically acceptable salts thereof can be administered via the parenteral, oral or rectal routes or by. . .

DETD . . . a pharmacodynamic study in db/db mice, the present inventors have shown that a single dose of a bisphenolic compound, i.e., **NDGA**, administered orally, reduced blood glucose level as early as 1.5 h after administration; the reduced blood glucose level was maintained. . .

DETD 6. EXAMPLE: HYPOGLYCEMIC ACTIVITY OF **NDGA**

DETD The following experiments demonstrate that the compounds described herein, for example **NDGA**, produce a significant and consistent hypoglycemic effect on **obese** diabetic mice, i.e., an art recognized model of diabetes mellitus. Further demonstrated are **NDGA**'s beneficial effects on glucose tolerance and the ability to stimulate glucose transport in adipocytes.

DETD This experiment illustrates the effectiveness of **NDGA** in reducing plasma glucose levels in **obese** diabetic db/db mice, a model recognized by those skilled in the art as being a representative model of non-insulin-dependent diabetes. . .

DETD Genetically altered **obese** diabetic mice (designated C57BL/Ks-db/db) were purchased from The Jackson Laboratory (Bar Harbor, Me., USA), and served as experimental animals. Male. . . at the start of the study. Diabetic mice designated C57BLIKS-db/db received, orally by gavage, daily for 2 days either vehicle, **NDGA** administered at 150 mg/kg bid (9:00 and 17:00), 250 and 350 mg/kg q.d., or metformin [250 mg (1510 mol)/kg q.d].. . .

DETD As shown in FIG. 1 and below in Table 1, oral administration of the **NDGA** to diabetic C57BL/Ks-db/db mice once daily for 2 days at dosage levels of 250 and 350 mg/kg resulted in dose. . . significant glucose reduction relative to vehicle (control) was observed after 3h at both dosage levels and 27h at 350 mg/kg. **NDGA** at 150 mg/kg twice daily also reduced plasma glucose levels. The change in body weight and overall food consumption values for mice treated with **NDGA** are shown in Table 2. No significant effects on the body weight and food consumption were observed with **NDGA** treatment up to 250 mg/kg q.d.

DETD . . . (mg/dL)

Treatment	(mg/kg)	3 h	P Value	27 h	P Value
Vehicle	--	-48.0	NA	-16.5	NA
Metformin	250 qd	-140.9	0.0009	-111.1	0.0013
NDGA	150 bid	-98.0	0.0647	-58.7	0.0597
NDGA	250 qd	-124.9	0.0052	-55.3	0.0634
NDGA	350 qd	-109.5	0.0147	-190.8	<0.0001

NA = nonapplicable

DETD . . . Change in Food Consumption

Treatment	Dose (mg/kg)	Body Weight (g) 24 h	(g/mouse) 0-24 h
Vehicle	--	0.1	6.1
Metformin	250 qd	-0.3	5.1
NDGA	150 qd	-0.1	5.4
NDGA	250 qd	-0.0	4.7
NDGA	350 qd	-0.6	3.6

DETD This experiment illustrates the effectiveness of **NDGA** in reducing plasma glucose levels in **obese** diabetic ob/ob mice, a model recognized by those skilled in the art as being a representative model of non-insulin-dependent diabetes. . .

DETD Genetically altered **obese** diabetic mice (designated C57BL/6J ob/ob) were purchased from The Jackson Laboratory (Bar Harbor, Me., USA), and served as experimental animals.. . .

DETD This example illustrates the beneficial effects of **NDGA** on glucose disposal.

DETD . . . as described in Section 6.1, above. Diabetic mice designated C57BLIKS-db/db received orally by gavage daily for 2 days, either vehicle, **NDGA** administered at 150 mg/kg bid (9:00 and 17:00), 250 and 350 mg/kg q.d., or metformin [250 mg (1510 mol)/kg q.d].. . .

DETD FIG. 3 and 4 show the effect of **NDGA** in the oral glucose tolerance test. **NDGA** at all dosage-levels significantly suppressed the

postprandial glucose levels at all timepoints after the glucose load. The oral glucose tolerance. . . was significantly improved as indicated by the area under the curve. This effect was dose dependent. These data indicate that **NDGA** enhances glucose utilization and improves the rate of glucose disposal in an animal model of insulin resistance, **obesity**, and NIDDM.

DETD This example illustrates the ability of **NDGA** to directly stimulate glucose transport in 3T3-L1 adipocytes, an 25 art recognized in vitro system that represents an important mode. . .

DETD . . . saline and switched to serum-free DMEM medium. Adipocytes were treated (in triplicate) for 18 hr with the indicated concentrations of **NDGA**. Concentrated stock solutions of **NDGA** were freshly prepared in dimethyl sulfoxide (DMSO) and diluted into culture medium. The final concentration of DMSO was 0.4% (v/v). . .

DETD **NDGA** increased the rate of basal glucose transport (i.e. no added insulin) in 3T3-L1 adipocytes by approximately 250% at 10 μM and by approximately 300% at 30 μM (FIG. 5). **NDGA** (10 and 30 μM) also sensitized the glucose transport system in adipocytes to subsequent stimulation with a sub-maximal concentration of insulin (0.5 nM). **NDGA** potentiated glucose transport in response to insulin by approximately 60% and 80% (FIG. 6). As would be recognized by those skilled in the art, these data indicate that the pure compound **NDGA** directly stimulates glucose transport in vitro, an effect that is consistent with the in vivo findings of enhanced glucose disposal. . .

CLM What is claimed is:

. . . A method for reducing blood glucose which comprises administering to a mammal a composition whose active ingredient consists essentially of **nordihydroguaiaretic acid** or a pharmaceutically acceptable salt thereof.

L9 ANSWER 2 OF 24 USPATFULL

Full Text

ACCESSION NUMBER: 2002:32531 USPATFULL

TITLE: Use of azalide antibiotic compositions for treating or preventing a bacterial or protozoal infection in mammals

INVENTOR(S): Canning, Peter Connor, Terre Haute, IN, UNITED STATES
Boettner, Wayne Alan, Noank, CT, UNITED STATES

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 2002019353	A1	20020214
APPLICATION INFO.:	US 2001-829672	A1	20010410 (9)

	NUMBER	DATE
PRIORITY INFORMATION:	US 2000-199961P	20000427 (60)

DOCUMENT TYPE: Utility

FILE SEGMENT: APPLICATION

LEGAL REPRESENTATIVE: Paul H. Ginsburg, Pfizer Inc., 235 East 42nd Street, 20th Floor, New York, NY, 10017-5755

NUMBER OF CLAIMS: 50

EXEMPLARY CLAIM: 1

LINE COUNT: 2922

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

AB Methods for treating or preventing bacterial or protozoal infections in mammals by administering a single dose of an antibiotic composition comprising a mixture of azalide isomers and a pharmaceutically acceptable vehicle are disclosed. Methods for increasing acute or chronic injection-site toleration in mammals by administering a single dose of antibiotic compositions comprising a mixture of azalide isomers and a pharmaceutically acceptable vehicle are also disclosed. A combination comprising: an antibiotic composition comprising a mixture of azalide isomers, a pharmaceutically acceptable carrier, and instructions for use in a single-dose administration is also disclosed.

L9 ANSWER 3 OF 24 CAPLUS COPYRIGHT 2002 ACS

Full Text

ACCESSION NUMBER: 1999:311102 CAPLUS

DOCUMENT NUMBER: 130:332910

TITLE: Methods and compositions for regulation of 5-alpha reductase activity

INVENTOR(S): Liao, Shutsung; Hiipakka, Richard A.

PATENT ASSIGNEE(S): Arch Development Corporation, USA

SOURCE: PCT Int. Appl., 48 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 9922728	A1	19990514	WO 1998-US23041	19981030
W: AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CU, CZ, DE, DK, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, UA, UG, US, UZ, VN, YU, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM				
RW: GH, GM, KE, LS, MW, SD, SZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG				
AU 9912898	A1	19990524	AU 1999-12898	19981030
EP 1027045	A1	20000816	EP 1998-956358	19981030
R: AT, BE, CH, DE, ES, FR, GB, GR, IT, LI, SE, PT, IE				

PRIORITY APPLN. INFO.: US 1997-63770P P 19971031
WO 1998-US23041 W 19981030
OTHER SOURCE(S): MARPAT 130:332910

AB Compds. that inhibit 5α-reductase are provided. The compds. are used to treat prostate cancer, breast cancer, **obesity**, skin disorders and baldness.

REFERENCE COUNT: 2 THERE ARE 2 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

AB Compds. that inhibit 5α-reductase are provided. The compds. are used to treat prostate cancer, breast cancer, **obesity**, skin disorders and baldness.

ST steroid 5alpha reductase inhibitor therapeutic; prostate cancer steroid 5alpha reductase inhibitor; breast cancer steroid 5alpha reductase inhibitor; **obesity** baldness steroid 5alpha reductase inhibitor; skin disorder steroid 5alpha reductase inhibitor

IT Alopecia
***Antiobesity agents
Skin, disease
(steroid 5-α reductase inhibitors, and therapeutic use)

IT 57-11-4, Octadecanoic acid, biological studies 58-27-5, Menadione 60-33-3, Linoleic acid, biological studies 72-48-0, Alizarin 81-54-9, Purpurin 81-64-1, Quinizarin 84-60-6, Anthraflavic acid 84-65-1, Anthraquinone 84-79-7, Lapachol 87-66-1, Pyrogallol 97-53-0, Eugenol 99-24-1, Methyl gallate 112-80-1, Oleic acid, biological studies 115-41-3, Pyrocatechol violet 117-12-4, Anthrarufin 117-39-5, Quercitin 120-80-9, 1,2-Benzenediol, biological studies 121-79-9, Propyl gallate 130-22-3 149-91-7, Gallic acid, biological studies 153-18-4, Rutin 301-00-8, α-Linolenic acid methyl ester 303-45-7, Gossypol 305-01-1, Esculetin 331-39-5, Caffeic acid 404-86-4, Capsaicin 446-72-0, Genistein 458-35-5 458-36-6, 4-Hydroxy-3-methoxycinnamaldehyde 458-37-7, Curcumin 463-40-1, α-Linolenic acid 476-66-4, Ellagic acid 480-16-0, Morin 480-18-2, Taxifolin 480-40-0, Chrysin 486-66-8, Daidzein 490-46-0, Epicatechin 491-67-8, Baicalein 491-80-5, Biochanin a **500-38-9**, **Nordihydroguaiaretic acid** 506-26-3, γ-Linolenic acid 517-28-2, Hematoxylin 520-18-3 525-82-6, Flavone 528-48-3, Fisetin 529-44-2, Myricetin 569-77-7, Purpurogallin 574-84-5, Fraxetin 577-33-3, Anthrarobin 604-59-1, α-Naphthoflavone 863-03-6, Epicatechin gallate 970-74-1, Epigallocatechin 989-51-5, Epigallocatechin gallate 1034-01-1, Octyl gallate 1080-12-2 1135-24-6, Ferulic acid 1138-60-9, Isopropyl gallate 1166-52-5, Dodecyl gallate 1927-04-4 1948-33-0 6051-87-2, β-Naphthoflavone 16326-32-2, γ-Linolenic acid methyl ester 16574-43-9, Bromopyrogallol red 25061-77-2 27876-94-4, Crocetin 32638-88-3, Pyrogallol red 36062-04-1, Tetrahydrocurcumin 51583-63-2 99518-16-8 104594-70-9, Caffeic acid phenethyl ester 127034-02-0, HZIV 82 173484-92-9, HZIV 75 183209-70-3, HZIV 145 220957-96-0, HZIV 90 224433-72-1 224433-76-5, HZIV 120 224433-78-7, HZIV 166 224433-86-7 224441-44-5, HZIV 160 224441-46-7, HZIV 134 224441-48-9, HZIV 92 224441-50-3, HZIV 142 224441-52-5, HZIV 68 224441-54-7, HZIV 63 224441-56-9, HZIV 169 224441-58-1, HZIV 74 224441-60-5, HZIV 144 224441-61-6, HZIV 168 224441-64-9, HZIV 107 224441-66-1, HZIV 148 224441-68-3, HZIV 109 224441-70-7, HZIV 165

RL: BAC (Biological activity or effector, except adverse); BSU (Biological study, unclassified); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(steroid 5-α reductase inhibitors, and therapeutic use)

L9 ANSWER 4 OF 24 CAPLUS COPYRIGHT 2002 ACS
Full Text
ACCESSION NUMBER: 1968:58408 CAPLUS
DOCUMENT NUMBER: 68:58408
TITLE: Inhibition of hamster caries by phenolic compounds
AUTHOR(S): Stralfors, Allan
CORPORATE SOURCE: Dental Fac., Univ. Umea, Umea, Swed.
SOURCE: Arch. Oral Biol. (1967), 12(12), 1375-85
CODEN: AOBIAR
DOCUMENT TYPE: Journal
LANGUAGE: English
AB Several phenols were tested for their effects on hamster dental caries. Quercitin, caffeic acid, and protocatechuic acid, at 0.2% of the diet, significantly inhibited dental caries by 32, 22, and 15%, resp., without decrease in body **wt. gain**. Coumarin and **nordihydroguaiaretic acid**, 0.2% of the diet, inhibited caries but significantly impaired growth. The inhibition of dental caries by the active substances is probably related to their inhibitory effect on oral microorganisms. 35 references.
IT 91-64-5 99-50-3 117-39-5 331-39-5 500-38-9
RL: BIOL (Biological study)
(teeth caries inhibition by)

L9 ANSWER 5 OF 24 CAPLUS COPYRIGHT 2002 ACS
Full Text
ACCESSION NUMBER: 2001:185516 CAPLUS
DOCUMENT NUMBER: 134:222060
TITLE: Compositions comprising conjugated linoleic acid (cla)
INVENTOR(S): Ghisalberti, Carlo
PATENT ASSIGNEE(S): Brazil
SOURCE: PCT Int. Appl., 28 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 3
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2001017374	A1	20010315	WO 2000-IB1277	20000908

W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CR, CU, CZ, DE, DK, DM, DZ, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG

PRIORITY APPLN. INFO.: IT 1999-MI1894 A 19990909
AB The present invention relates to new oral compns. comprising CLA in combination with food grade antioxidants and the use of said combination for the manuf. of a dietetic compn. or a medicament useful in the treatment of atherosclerosis, **overweight** and in enhancing the immune response.
REFERENCE COUNT: 7 THERE ARE 7 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT
IT **Antiobesity** agents
Beverages
Feed additives
Food additives
Immunostimulants
Orange juice
Oxidative stress, biological
(compns. comprising conjugated linoleic acid for dietetic foods and medicaments)
IT 58-95-7, Vitamin E acetate 59-02-9, α-Tocopherol 89-65-6, Erythorbic acid 89-65-6D, Erythorbic acid, salts 91-53-2, Ethoxyquin 117-39-5, Quercetin 121-79-9, Propyl gallate 149-91-7D, Gallic acid, esters 153-18-4 303-98-0, Coenzyme Q10 476-70-0, Boldine

500-38-9, Nordihydroguaiaretic acid
500-38-9D, Nordihydroguaiaretic acid, esters
502-65-8, Lycopene 520-26-3, Hesperidin 1200-22-2, α-Lipoic acid 1421-63-2 1839-11-8, 9,11-Octadecadienoic acid 6381-77-7, Sodium erythorbate 9005-37-2, Propylene glycol alginate 10597-60-1 11034-77-8, Anacardic acid 12738-23-7, Oryzanol 22880-03-1, 10,12-Octadecadienoic acid 32619-42-4, Oleuropeine 37330-39-5, Cardanol 57486-25-6, Cardol 57828-26-9D, Lipoic acid, derivs. 61276-17-3, Verbascoside 121250-47-3, Conjugated linoleic acid 168131-31-5, 11,13-Octadecadienoic acid 218607-75-1D, Octadecadienoic acid, derivs.
RL: FFD (Food or feed use); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(compns. comprising conjugated linoleic acid for dietetic foods and medicaments)

=> d ibib abs kwic 6-10

L9 ANSWER 6 OF 24 USPATFULL
Full Text

ACCESSION NUMBER:	88:62483 USPATFULL
TITLE:	Modification of plant extracts from zygophyllaceae and pharmaceutical use therefor
INVENTOR(S):	Jordan, Russell T., Fort Collins, CO, United States
PATENT ASSIGNEE(S):	Chemex Pharmaceuticals, Inc., Denver, CO, United States (U.S. corporation)

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 4774229		19880927
APPLICATION INFO.:	US 1986-860654		19860507 (6)

RELATED APPLN. INFO.:	Continuation of Ser. No. US 1982-365784, filed on 5 Apr 1982, now abandoned which is a continuation-in-part of Ser. No. US 1979-49886, filed on 19 Jun 1979, now abandoned
DOCUMENT TYPE:	Utility
FILE SEGMENT:	Granted
PRIMARY EXAMINER:	Rollins, John
LEGAL REPRESENTATIVE:	Kenyon & Kenyon
NUMBER OF CLAIMS:	21
EXEMPLARY CLAIM:	1
LINE COUNT:	835

CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB A mixture of an extract from a plant belonging to the Zygophyllaceae family containing phenolic compositions and a nonalkali metal salt is useful as a pharmaceutical agent, for example, in the treatment of cancer, nonmalignant tumors, osteomyelitis, psoriasis and warts.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.
SUMM . . . anticancer activity have concluded that the tea is not beneficial in the treatment of cancer. Extracts from Larrea plants, including **nordihydroguaiaretic acid** (**NDGA**), have also been investigated for potential antibacterial activity and have been found to possess such activity in vitro. Additionally, phenolic. . .
SUMM . . . are contained in the extracts of the mixtures of the present invention include, guaiacol, guaiaconic acid and lignans such as: **nordihydroguaiaretic acid**, guaiaretic acid, norisoguaiacin, 3'-demethoxyisoguaiacin, dihydroguaiaretic acid, partially demethylated dihydroguaiaretic acid, 1-(4(or 3)-hydroxyphenyl)-6,7 dihydroxy-2,3-dimethyl-1,2,3,4-tetrahydronaphthalene, 1-(3,4-dihydroxyphenyl)-6,7-dihydroxy-2,3-dimethyl 1,2,3,4tetrahydronaphthalene, 1-(3,4-dihydroxyphenyl)-2,3-dimethyl-4-(4(or 3)-hydroxyphenyl) butane, 1-(3,4-dihydroxyphenyl)-2,3-dimethyl-4-(3,4,5-trihydroxy-phenyl) butane,. . .
SUMM Preferred extracts are those which contain one or more of the following phenolic compositions: guaiacol, **nordihydroguaiaretic acid**, guaiaretic acid, norisoguaiacin, 3'-demethoxyisoguaiacin, dihydroguaiaretic acid, partially demethylated dihydroguaiaretic acid, 1,4-bis(3(or 4)-hydroxy-4(or 3)-methoxyphenyl) butane, 1-(3,4-dihydroxphenyl-4-(3(or 4)-methoxy-4(or 3)-hydroxyphenyl) butane, 14(or 3)hydroxyphenyl)-. . .
DETD The catechol content was determined by the method of Duisberg, P. C., et al. "Determination of **Nordihydroguaiaretic Acid** in the Leaf of Larrea divaricata", Anal. Chem., 21:1393-96, which is incorporated

herein as a reference.

DETD . . . their tumors treated topically with an ointment comprised of about 54 grams of zinc chloride, 5 grams quercetin, 10 grams **NDGA**, about 10 grams ascorbic acid, about 20 grams of water and about 8 grams of polyethylene glycol.

DETD . . . 64 year old male Caucasian with adenocarcinoma of the right lung was treated with a preparation comprised of zinc chloride, **NDGA** and ascorbic acid. The preparation was prepared by blending to obtain a homogeneous mixture 54 grams of zinc chloride, about 28 grams of water, about 11 grams of **NDGA** and about 7 grams of ascorbic acid. Capsules were prepared by placing 250 milligrams of the preparation in each capsule.. . .

DETD . . . treated with a preparation comprising about 55 grams of zinc chloride, about 5 grams of quercetin, about 1 gram of **NDGA**, about 1 gram of ascorbic acid and 20 grams of water all formulated into about 15 grams of a base. . .

DETD . . . clinically improving and X-rays indicated that the cancer was decreasing. By 7 weeks after treatment the dog had experienced a **weight gain** of 7 kg, had only a slight limp and was increasing its exercise. About 9 weeks after the initial administration,. . .

CLM What is claimed is:

. . . claim 1 wherein the extract contains at least one phenolic composition selected from the group consisting of guaiacol; guaiaconic acid; **nordihydroguaiaretic acid**; guaiaretic acid; norisoguaiacin; 3'-demethoxyisoguaiacin; dihydroguaiaretic acid; partially demethylated dihydroguaiaretic acid; 1-(4-hydroxyphenyl)-6,7-dihydroxy-2,3-dimethyl-1,2,3,4,-tetrahydronaphthalene; 1-(3-hydroxyphenyl)-6,7-dihydroxy-2,3-dimethyl-1,2,3,4-tetrahydronaphthalene; 1-(3,4-dihydroxyphenol)6,7-dihydroxy-2,3-dimethyl-1,2,3,4-tetrahydronaphthalene; 1-(3,4-dihydroxyphenyl)-2,3-dimethyl-4-(3-hydroxyphenyl) butane; 1-(3,4-dihydroxyphenyl)-2,3-dimethyl-4-(4-hydroxyphenyl) butane; 1-(3,4-dihydroxyphenyl)-2,3-dimethyl-4-(3,4,5-trihydroxyphenyl) butane; 1-(3,4-dihydroxyphenyl)-2,3-dimethyl-4-[3-(4-hydroxy-3-methylbenzyloxy)-4-hydroxyphenyl]. . .

. . . of the mixture and the plant extract contains at least one phenolic composition selected from the gorup consisting of guaiacol; **nordihydroguaiaretic acid**; guaiaretic acid; norisoguaiacin; 3'-demethoxyisoguaiacin; dihydroguaiaretic acid; partially demethylated dihydroguaiaretic acid; 1,4-bis (3-hydroxy-4-methoxyphenyl) butane; 1,4-bis(4-hydroxy-3-methoxyphenyl) butane; 1-(3-4-dihydroxyphenyl)-4-(3-methoxy-4-hydroxyphenyl) butane; 1-(3,4-dihydroxyphenyl)-4-(4-methoxy-3-hydroxyphenyl) 4-tetrahydronaphthalen; 1-(3-hydroxyphenyl)-6,7-dihydroxy-2,. . .

. . . which comprises topically administering to a mammal in need of said treatment a composition containing a pharmacologically active amount of **nordihydroguaiaretic acid** and a nonalkali metal halide selected from the group consisting of copper, manganese, cadmium, antimony and zinc.

. . . skin comprising topically administering to a mammal in need of said treatment a composition containing a pharmacologically active amount of **nordihydroguaiaretic acid** and a zinc metal salt.

. . . comprising the topical administration to a mammal in need of said treatment a composition containing a pharmacologically active amount of **nordihydroguaiaretic acid** and a zinc metal salt.

18. A composition comprising **nordihydroguaiaretic acid** and a nonalkali metal halide selected from the group consisting of copper, manganese, cadmium, antimony and zinc.

L9 ANSWER 7 OF 24 CAPLUS COPYRIGHT 2002 ACS
Full Text

ACCESSION NUMBER: 1970:511213 CAPLUS
DOCUMENT NUMBER: 73:111213
TITLE: Effect of antioxidants in the autoxidation of methyl conjugated cis,trans-octadecadienoates
AUTHOR(S): Fukuzumi, Kazuo; Ikeda, Nobuo
CORPORATE SOURCE: Fac. Eng., Nagoya Univ., Nagoya, Japan
SOURCE: J. Amer. Oil Chem. Soc. (1970), 47(10), 369-70
CODEN: JAOCA7
DOCUMENT TYPE: Journal
LANGUAGE: English

AB The effect of antioxidants on the autoxidn. of Me conjugated cis,trans-octadecadienoates was evaluated by estg. the induction period by

measuring the increase in wt. with time. Peroxide values and mol. wts. were also used to det. extent of oxidn. Uv and ir absorption were measured to det. conjugated dienes and isolated trans double bonds. Antioxidants, such as butylated hydroxyanisole (BHA), butylated hydroxytoluene (BHT), Pr gallate (PG), and sesamol, lengthened the induction period as much as 7-12 times. After autoxidn. to a wt. gain of 10 mg per 1.5 g, the antioxidant contg. samples had higher mol. wts. and lower diene contents that the control samples. The induction periods were shorter and the peroxide values lower, with or without antioxidants, for the conjugated dienoates than for the nonconjugated dienoates. Effect of antioxidants might be explained by the formation of an H bond of the OH of the antioxidant and π-electrons as well as the inhibition of the chain reaction.

IT 51-48-9, uses and miscellaneous 59-02-9 118-82-1 121-79-9 128-37-0 **500-38-9** 533-31-3 25013-16-5
RL: USES (Uses)
(antioxidants, for methyl octadecadienoate)

L9 ANSWER 8 OF 24 USPATFULL
Full Text

ACCESSION NUMBER:	2001:119278 USPATFULL
TITLE:	Human leukocyte 12-lipoxygenase and its role in the pathogenesis of disease states
INVENTOR(S):	Nadler, Jerry L., La Crescenta, CA, United States Natarajan, Rama, Hacienda Heights, CA, United States

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 2001009900	A1	20010726
APPLICATION INFO.:	US 2000-739843	A1	20001220 (9)

RELATED APPLN. INFO.:	Division of Ser. No. US 1997-945744, filed on 3 Nov 1997, GRANTED, Pat. No. US 6191169 Continuation-in-part of Ser. No. WO 1996-US6328, filed on 3 May 1996, UNKNOWN Continuation-in-part of Ser. No. US 1995-434681, filed on 4 May 1995, ABANDONED Continuation-in-part of Ser. No. WO 1994-US89, filed on 4 Jan 1994, UNKNOWN Continuation-in-part of Ser. No. US 1992-936660, filed on 28 Aug 1992, ABANDONED
DOCUMENT TYPE:	Utility
FILE SEGMENT:	APPLICATION
LEGAL REPRESENTATIVE:	ROTHWELL, FIGG, ERNST & MANBECK, P.C., 555 13TH STREET, N.W., SUITE 701, EAST TOWER, WASHINGTON, DC, 20004
NUMBER OF CLAIMS:	38
EXEMPLARY CLAIM:	1
NUMBER OF DRAWINGS:	24 Drawing Page(s)
LINE COUNT:	1653

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

AB The present invention relates to a method for inhibiting the etiology of disease in patients having a disease state caused by an excess of 12-lipoxygenase or its products. In particular, the invention provides for administration of a human leukocyte 12-lipoxygenase pathway inhibitor to inhibit disease etiology, to inhibit the proliferation of breast cancer and to increase insulin receptor phosphorylation in a patient having Type II diabetics.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

DETD . . . In addition, the proliferation of MCF-7 cells was significantly inhibited by three LO inhibitors, baicalein (10 μM), CDC (10-5M) and **NDGA** (30 μM), but not by a cyclooxygenase inhibitor, ibuprofen (10-5M). Treatment of serum-starved MCF-7 cells with EGF for four hours. . .

DETD [0121] 2. Evidence that 12-LO mRNA expression progressively increases in rat pancreatic islets from lean non-diabetic animals, to **obese** pre-diabetic and **obese** diabetic animals (see FIG. 21).

DETD [0123] 4. In vivo data (see Table 2) that urinary 12-HETE levels are much higher in male diabetic **obese** ZDF rats (a model of NIDDM) compared to lean ZDF non-diabetic rats. Interestingly, **obese** female ZDF rats which are phenotypically like pre-diabetic humans show intermediate levels of 12-HETE in urine.

TABLE 2

Urinary 12-HETE in ZDF Rats

	pg/total urine vol.

Diabetic Male	Obese Ctrl	2022 ± 372
Non-diabetic	Female Obese Ctrl	1007
	Female Obese Mg2+	86
Non-diabetic	Lean Male	--
	Lean Female	--

DETD . . . rats (lane 11 vs lane 21 in FIG. 22). Interestingly, 12-LO mRNA levels in skeletal muscle are also higher in obese female ZDF rats that are prone to get diabetes. In this figure, the 312 bp band is the 12-LO band. . .

DETD . . . expression in soleus muscle was over 5-fold greater in GK vs Wistar rats (0.6±0.1 Wistar vs 3.1±0.76 GK). The ZDF obese rats demonstrated an increase in blood glucose concentration and weight compared to the ZDF lean controls (558±75 vs 170±5 mg/dl. . . quadriceps muscle in the diabetic and lean ZDF rats. 12-LO mRNA expression was increased by 4-7 fold in the diabetic obese ZDF rats compared to the lean ZDF controls. 12-LO protein expression was similarly increased in heart tissue (5-fold) in the. . . ZDF vs the lean ZDF controls. These data reflect that muscle 12-LO expression is markedly increased in both lean and obese rat models of NIDDM.

DETD . . . expression and 12-HETE levels. High Mg diets (Purina 5008 diet containing 1% Mg) were fed to one group of ZDF obese (diabetic fatty) male rats while control diets (Purina 5008 plus 0.2% Mg) were fed to another group. As can be. . . 12-HETE Excretion Rate in ZDF Rat Models

ZDF PAT	12-HETE (pg/mm)
ZDF lean (n = 4)	a0.4 ± 0.07
ZDF obese (n = 6)	6.12 ± 1.2
ZDF obese (n = 6) with h.Mg diet	b3.72 ± 0.5

Values are mean ± SE.
n is the number of rats.
Values in ZDF lean group and ZDF obese with high magnesium diet group are different from ZDF obese group at
ap < 0.001 and
bp < 0.05 respectively.

DETD [0136] Values are mean±SE. n is the number of rats. values in ZDF lean group and ZDF obese with high magnesium diet group are different from ZDF obese group at ap<0.001 and bp<0.05 respectively.

CLM What is claimed is:

9. The method of claim 6, in which said inhibitor is NDGA, CDC, panaxynol, biacalein, pioglitazone, aminoguanidine or a ribozyme which cleaves hl 12-lipoxygenase mRNA.

14. The method of claims 10, 11, 12 or 13, wherein said inhibitor is NDGA, CDC, panaxynol, baicalein, pioglitazone, aminoguanidine or a ribozyme which cleaves hi 12-lipoxygenase mRNA.

23. The method of claim 22 in which said human 12-lipoxygenase inhibitor is NDGA, CDC, panaxynol, baicalein, pioglitazone, aminoguanidine or a ribozyme which cleaves hl 12-lipoxygenase mRNA.

26. The method of claim 25 in which said human 12-lipoxygenase inhibitor is NDGA, CDC, panaxynol, baicalein, pioglitazone, aminoguanidine or a ribozyme which cleaves hl 12-lipoxygenase mRNA.

L9 ANSWER 9 OF 24 USPATFULL
Full Text

ACCESSION NUMBER:	2000:70810 USPATFULL
TITLE:	Use of IGF-I for the treatment of renal insufficiencies, steriod toxicity and related indications
INVENTOR(S):	Acott, Philip D., Halifax, Canada Crocker, John F. S., Halifax, Canada
PATENT ASSIGNEE(S):	Dalhousie University, Halifax, Canada (non-U.S. corporation)

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 6071880		20000606
APPLICATION INFO.:	US 1999-307005		19990507 (9)
RELATED APPLN. INFO.:	Division of Ser. No. US 1997-933196, filed on 16 Sep 1997, now patented, Pat. No. US 5985830 which is a		

continuation-in-part of Ser. No. US 1996-710331, filed on 16 Sep 1996, now abandoned
DOCUMENT TYPE: Utility
FILE SEGMENT: Granted
PRIMARY EXAMINER: Henley, III, Raymond
LEGAL REPRESENTATIVE: Gray Cary Ware & Freidenrich, Reiter, Stephen E.
NUMBER OF CLAIMS: 9
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 5 Drawing Figure(s); 5 Drawing Page(s)
LINE COUNT: 1306
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB In accordance with the present invention, there are provided methods for the treatment of chronic renal insufficiencies and related chronic indications in mammals, employing IGF-I as the active agent. In accordance with one embodiment of the present invention, it has been discovered that IGF-I is an effective agent for enhancing kidney development in mammals suffering from chronic organ injury. In accordance with a further embodiment of the present invention, it has been discovered that IGF-I is an effective agent for protecting prepubescent subjects, such as prepubescent mammals and neonates, from the ongoing toxicity of treatment with steroid hormones. In accordance with a still further embodiment of the present invention, it has been discovered that IGF-I is an effective agent for maintaining substantially normal growth in neonates and pre-pubescent mammals exposed to high dose steroid hormone therapy.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.
SUMM . . . Eds. Gardner KD, Bernstein J, 55-98 (1990)). Reproducible models of PKD include those induced by organic chemicals--specifically diphenylamine, diphenylthiazole and **nordihydroguaiaretic acid** and those induced by the administration of glucocorticoids (Avner et al., supra) and (Perey et al., Science, 158:494-496 (1967)).
DETD . . . A general regression methodology for ROC curve estimation. J Royal Stat Soc (Series B) 42:109-142 (1980)). Difference in overall body **weight gain** was assessed using the Tukey-Kramer method of pairwise comparisons of means. Pairwise comparisons of the mean glucose concentrations were done. . .
DETD . . . the reduction of the glucocorticoid-induced catabolism and weight loss. The control group, IGF-I, and MPA+IGF-I group showed no difference in **weight gain** and were statistically different than the MPA treated group alone.
DETD There was also observed to be an improvement of the poor **weight gain** secondary to glucocorticoid-induced catabolism in the IGF-I treated group of the GIPKD model. Pairwise comparisons (Tukey-Kramer) of the 5 day. . . and the MPA+IGF-I litters (1.075±0.105, n=4 litters) was not different (p=0.177), although control was different from MPA+IGF-I (p=0.007). However, the **weight gain** per mouse of the MPA litters (0.583 g±0.086, n=6 litters) was significantly smaller than the other groups (all three p<0.010). For this experiment, these multiple pairwise comparisons infer a ranking of the total **weight gain**: Control<IGF-I<MPA+IGF-I>MPA. These experiments clearly demonstrate that IGF-I reverses the negative glucocorticoid effect on **weight gain** in the immediate postnatal period.
DETD . . . In addition, IGF-I reduces glucocorticoid-induced catabolism and weight loss. The control group, IGF-I, and MPA+IGF-I group showed no difference in **weight gain**; this profile was statistically different than the poor **weight gain** of the MPA treated group. The IGF-I+MPA treated animals had lower creatinine (see Table 1), urea (see Table 1), and. . .
DETD . . . at 24 hours of age have >80% mortality within 10 days of age, with significant evidence of catabolism and poor **weight gain**. All treated mice die by 13 days of age. In the group which received IGF-I in addition to MPA, there. . .
DETD . . . an 88% mortality within 10 days of age (i.e., 108 of 123 died), with significant evidence of catabolism and poor **weight gain**. In the group receiving IGF-I in addition to methylprednisolone acetate, there was a significant increase in growth and a reduction. . .

L9 ANSWER 10 OF 24 USPATFULL
Full Text
ACCESSION NUMBER: 1999:146529 USPATFULL
TITLE: Use of IGF-I for the treatment of kidney disorders
INVENTOR(S): Acott, Philip D., Halifax, Canada
Crocker, John F. S., Halifax, Canada
PATENT ASSIGNEE(S): Dalhousie University, Halifax, Canada (non-U.S.

corporation)

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 5985830		19991116
APPLICATION INFO.:	US 1997-933196		19970916 (8)

RELATED APPLN. INFO.: Continuation of Ser. No. US 1996-710331, filed on 16 Sep 1996, now abandoned
DOCUMENT TYPE: Utility
FILE SEGMENT: Granted
PRIMARY EXAMINER: Henley, III, Raymond
LEGAL REPRESENTATIVE: Gray Cary Ware & Freidenrich, Reiter, Stephen E., Learn, June M.
NUMBER OF CLAIMS: 24
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 5 Drawing Figure(s); 5 Drawing Page(s)
LINE COUNT: 1205
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

AB In accordance with the present invention, there are provided methods for the treatment of polycystic kidney disease and related indications in mammals, employing IGF-I as the active agent. In accordance with another embodiment of the present invention, it has been discovered that IGF-I is an effective agent for the treatment of renal dysplasias and/or renal hypoplasias in mammals. In accordance with yet another embodiment of the present invention, it has been discovered that IGF-I is an effective agent for enhancing glomerular development in mammals. In accordance with still another embodiment of the present invention, it has been discovered that IGF-I is an effective agent for enhancing kidney development in mammals suffering from chronic organ injury.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

SUMM . . . Gardner K D, Bernstein J, 55-98 (1990)). Reproducible models of PKD include those induced by organic chemicals--specifically diphenylamine, diphenylthiazole and **nordihydroguaiaretic acid** and those induced by the administration of glucocorticoids (Avner et al., supra) and (Perey et al., Science, 158: 494-496 (1967)).

DETD . . . A general regression methodology for ROC curve estimation. J Royal Stat Soc (Series B) 42:109-142 (1980)). Difference in overall body **weight gain** was assessed using the Tukey-Kramer method of pairwise comparisons of means. Pairwise comparisons of the mean glucose concentrations were done. . .

DETD . . . the reduction of the glucocorticoid-induced catabolism and weight loss. The control group, IGF-I, and MPA+IGF-I group showed no difference in **weight gain** and were statistically different than the MPA treated group alone.

DETD There was also observed to be an improvement of the poor **weight gain** secondary to glucocorticoid-induced catabolism in the IGF-I treated group of the GIPKD model. Pairwise comparisons (Tukey-Kramer) of the 5 day. . . and the MPA+IGF-I litters (1.075±0.105, n=4 litters) was not different (p=0.177), although control was different from MPA+IGF-I (p=0.007). However, the **weight gain** per mouse of the MPA litters (0.583g (0.086, n=6 litters) was significantly smaller than the other groups (all three p<0.010). For this experiment, these multiple pairwise comparisons infer a ranking of the total **weight gain**: Control≥IGF-I≥MPA+IGF-I>MPA. These experiments clearly demonstrate that IGF-I reverses the negative glucocorticoid effect on **weight gain** in the immediate postnatal period.

DETD . . . In addition, IGF-I reduces glucocorticoid-induced catabolism and weight loss. The control group, IGF-I, and MPA+IGF-I group showed no difference in **weight gain**; this profile was statistically different than the poor **weight gain** of the MPA treated group. The IGF-I+MPA treated animals had lower creatinine (see Table 1), urea (see Table 1), and. . .

DETD . . . at 24 hours of age have >80% mortality within 10 days of age, with significant evidence of catabolism and poor **weight gain**. All treated mice die by 13 days of age. In the group which received IGF-I in addition to MPA, there. . .

DETD . . . an 88% mortality within 10 days of age (i.e., 108 of 123 died), with significant evidence of catabolism and poor **weight gain**. In the group receiving IGF-I in addition to methylprednisolone acetate, there was a significant increase in growth and a reduction. . .

=> d ibib abs kwic 11-15

L9 ANSWER 11 OF 24 CAPLUS COPYRIGHT 2002 ACS
Full Text
ACCESSION NUMBER: 1982:198039 CAPLUS
DOCUMENT NUMBER: 96:198039
TITLE: Safety evaluation of the estimated daily intake of food additives
AUTHOR(S): Taniguchi, Shigeru; Ohgaki, Sumiko; Yamada, Akio; Morita, Shigeru; Noda, Tsutomu
CORPORATE SOURCE: Osaka City Inst. Public Health Environ. Sci., Osaka, 543, Japan
SOURCE: Shokuhin Eiseigaku Zasshi (1982), 23(1), 1-20
CODEN: SKEZAP; ISSN: 0015-6426
DOCUMENT TYPE: Journal
LANGUAGE: Japanese
AB Rats were fed diets contg. 13 food additives (primarily antioxidants and preservatives) at dose levels of 1, 10, and 100 fold the estd. daily intake by Japanese for 1, 3, and 12 mo. An increase of liver wt. was obsd. at the medium dose level. The group receiving the highest dose showed decreases in hematocrit value and body wt. gain (female) and an increase in liver wt., mainly due to hyperplasia. After 12 mo, fatty degeneration of the liver occurred in females given the highest dose. It is likely that these pathol. changes were caused by the synergistic effects of the food additives tested.
IT 92-52-4, biological studies 94-26-8 121-79-9 128-44-9 137-40-6 500-38-9 532-32-1 4418-26-2 24634-61-5 25013-16-5 30587-81-6
RL: PRP (Properties)
(toxicity of, to liver, in food additive mixt.)

L9 ANSWER 12 OF 24 USPATFULL
Full Text
ACCESSION NUMBER: 2001:25934 USPATFULL
TITLE: Human leukocyte 12-lipoxygenase and its role in the pathogenesis of disease states
INVENTOR(S): Nadler, Jerry L., La Crescenta, CA, United States
Natarajan, Rama, Hacienda Heights, CA, United States
PATENT ASSIGNEE(S): City of Hope, Duarte, CA, United States (U.S. corporation)

	NUMBER	KIND	DATE	
PATENT INFORMATION:	US 6191169	B1	20010220	
	WO 9634943		19961107	
APPLICATION INFO.:	US 1997-945744		19971103	(8)
	WO 1996-US6328		19960503	
			19971103	PCT 371 date
			19971103	PCT 102(e) date

RELATED APPLN. INFO.: Continuation-in-part of Ser. No. US 1995-434681, filed on 4 May 1995, now abandoned Continuation-in-part of Ser. No. WO 1994-US89, filed on 4 Jan 1994 Continuation-in-part of Ser. No. US 1992-936660, filed on 28 Aug 1992, now abandoned
DOCUMENT TYPE: Utility
FILE SEGMENT: Granted
PRIMARY EXAMINER: Criares, Theodore J.
LEGAL REPRESENTATIVE: Rothwell, Figg, Ernst & Manbeck
NUMBER OF CLAIMS: 15
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 38 Drawing Figure(s); 24 Drawing Page(s)
LINE COUNT: 1665
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB The present invention relates to a method for inhibiting the etiology of disease in patients having a disease state caused by an excess of 12-lipoxygenase or its products. In particular, the invention provides for administration of a human leukocyte 12-lipoxygenase pathway inhibitor to inhibit disease etiology, to inhibit the proliferation of breast cancer and to increase insulin receptor phosphorylation in a patient having Type II diabetics.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.
DETD . . . addition, the proliferation of MCF-7 cells was significantly inhibited by three LO inhibitors, baicalein (10 μM), CDC (10--5 M) and NDGA (30 μM), but not by a cyclooxygenase inhibitor, ibuprofen (10-5 M). Treatment of serum-starved MCF-7 cells with EGF for four. . .

DETD 2. Evidence that 12-LO mRNA expression progressively increases in rat pancreatic islets from lean non-diabetic animals, to **obese** pre-diabetic and **obese** diabetic animals (see FIG. 21).

DETD 4. In vivo data (see Table 2) that urinary 12-HETE levels are much higher in male diabetic **obese** ZDF rats (a model of NIDDM) compared to lean ZDF non-diabetic rats. Interestingly, **obese** female ZDF rats which are phenotypically like pre-diabetic humans show intermediate levels of 12-HETE in urine.

DETD TABLE 2

Urinary 12-HETE in ZDF Rats

	pg/total urine vol.
Diabetic Male **Obese** Ctrl	2022 ± 372
Non-diabetic	
Female **Obese** Ctrl	1007
Female **Obese** Mg2+	86
Non-diabetic	
Lean Male	--
Lean Female	--

DETD . . . rats (lane 11 vs lane 21 in FIG. 22). Interestingly, 12-LO mRNA levels in skeletal muscle are also higher in **obese** female ZDF rats that are prone to get diabetes. In this figure, the 312 bp band is the 12-LO band. . .

DETD . . . expression in soleus muscle was over 5-fold greater in GK vs Wistar rats (0.6±0.1 Wistar vs 3.1±0.76 GK). The ZDF **obese** rats demonstrated an increase in blood glucose concentration and weight compared to the ZDF lean controls (558±75 vs 170±5 mg/dl. . . quadriceps muscle in the diabetic and lean ZDF rats. 12-LO mRNA expression was increased by 4-7 fold in the diabetic **obese** ZDF rats compared to the lean ZDF controls. 12-LO protein expression was similarly increased in heart tissue (5-fold) in the. . . ZDF vs the lean ZDF controls. These data reflect that muscle 12-LO expression is markedly increased in both lean and **obese** rat models of NIDDM.

DETD . . . expression and 12-HETE levels. High Mg diets (Purina 5008 diet containing 1% Mg) were fed to one group of ZDF **obese** (diabetic fatty) male rats while control diets (Purina 5008 plus 0.2% Mg) were fed to another group. As can be. . .

DETD . . . 12-HETE Excretion Rate in ZDF Rat Models

ZDF RAT	12-HETE (pg/min)
ZDF lean (n = 4)	a 0.4 ± 0.07
ZDF **obese** (n = 6)	6.12 ± 1.2
ZDF **obese** (n = 6) with h.Mg diet	b 3.72 ± 0.5

Values are mean ± SE. n is the number of rats. Values in ZDF lean group and ZDF **obese** with high magnesium diet group are different from ZDF **obese** group at a $p < 0.001$ and b $p < 0.05$ respectively.

CLM What is claimed is:

4. The method of claim 1, in which said hl 12-lipoxygenase pathway inhibitor is **NDGA**, CDC, panaxynol, baicalein or a ribozyme which cleaves hl 12-lipoxygenase mRNA.

L9 ANSWER 13 OF 24 MEDLINE

Full Text

ACCESSION NUMBER: 1999107847 MEDLINE

DOCUMENT NUMBER: 99107847 PubMed ID: 9890957

TITLE: Glucose decreases Na+,K+-ATPase activity in pancreatic beta-cells. An effect mediated via Ca2+-independent phospholipase A2 and protein kinase C-dependent phosphorylation of the alpha-subunit.

AUTHOR: Owada S; Larsson O; Arkhammar P; Katz A I; Chibalin A V; Berggren P O; Bertorello A M

CORPORATE SOURCE: Rolf Luft Center for Diabetes Research L6B:01, Department of Molecular Medicine, Karolinska Institutet, Karolinska Hospital, S-171 76 Stockholm, Sweden.

SOURCE: JOURNAL OF BIOLOGICAL CHEMISTRY, (1999 Jan 22) 274 (4) 2000-8.
Journal code: HIV; 2985121R. ISSN: 0021-9258.

PUB. COUNTRY: United States
Journal; Article; (JOURNAL ARTICLE)

LANGUAGE: English

FILE SEGMENT: Priority Journals

ENTRY MONTH: 199902

ENTRY DATE: Entered STN: 19990301
Last Updated on STN: 19990301

Entered Medline: 19990216

AB In the pancreatic beta-cell, glucose-induced membrane depolarization promotes opening of voltage-gated L-type Ca2+ channels, an increase in cytoplasmic free Ca2+ concentration ([Ca2+]i), and exocytosis of insulin. Inhibition of Na+,K+-ATPase activity by ouabain leads to beta-cell membrane depolarization and Ca2+ influx. Because glucose-induced beta-cell membrane depolarization cannot be attributed solely to closure of ATP-regulated K+ channels, we investigated whether glucose regulates other transport proteins, such as the Na+,K+-ATPase. Glucose inhibited Na+,K+-ATPase activity in single pancreatic islets and intact beta-cells. This effect was reversible and required glucose metabolism. The inhibitory action of glucose was blocked by pretreatment of the islets with a selective inhibitor of a Ca2+-independent phospholipase A2. Arachidonic acid, the hydrolytic product of this phospholipase A2, also inhibited Na+, K+-ATPase activity. This effect, like that of glucose, was blocked by **nordihydroguaiaretic acid**, a selective inhibitor of the lipooxygenase metabolic pathway, but not by inhibitors of the cyclooxygenase or cytochrome P450-monooxygenase pathways. The lipooxygenase product 12(S)-HETE (12-S-hydroxyeicosatetranoic acid) inhibited Na+,K+-ATPase activity, and this effect, as well as that of glucose, was blocked by bisindolylmaleimide, a specific protein kinase C inhibitor. Moreover, glucose increased the state of alpha-subunit phosphorylation by a protein kinase C-dependent process. These results demonstrate that glucose inhibits Na+, K+-ATPase activity in beta-cells by activating a distinct intracellular signaling network. Inhibition of Na+,K+-ATPase activity may thus be part of the mechanisms whereby glucose promotes membrane depolarization, an increase in [Ca2+]i, and thereby insulin secretion in the pancreatic beta-cell.

CT . . .

pharmacology

*Islets of Langerhans: DE, drug effects
Islets of Langerhans: EN, enzymology
Islets of Langerhans: PH, physiology
Membrane Potentials
Mice
Mice, Obese
*Na(+)-K(+)-Exchanging ATPase: AI, antagonists & inhibitors
*Phospholipases A: ME, metabolism
Phosphorylation
*Protein Kinase C: ME, metabolism

L9 ANSWER 14 OF 24 USPATFULL

Full Text

ACCESSION NUMBER: 2000:174702 USPATFULL
TITLE: Taste enhancing food additives
INVENTOR(S): Gilbertson, Timothy A., St. Amant, LA, United States
PATENT ASSIGNEE(S): Board of Supervisors of Louisiana State University and Agricultural and Mechanical College, Baton Rouge, LA, United States (U.S. state government)

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 6166076		20001226
APPLICATION INFO.:	US 1997-987494		19971209 (8)

	NUMBER	DATE
PRIORITY INFORMATION:	US 1996-88355P	19961213 (60)

DOCUMENT TYPE: Utility
FILE SEGMENT: Granted
PRIMARY EXAMINER: Criares, Theodore J.
LEGAL REPRESENTATIVE: Davis, Bonnie J., Runnels, John H.
NUMBER OF CLAIMS: 20
EXEMPLARY CLAIM: 1
LINE COUNT: 676
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

AB Cis-polyunsaturated fatty acids stimulate taste receptor cells in the mouth. The addition of free fatty acids to foods, such as low calorie fat substitutes or sugar substitutes, can make the foods more palatable. Certain ion channels in taste receptor cells are directly sensitive to extracellular applications of free fatty acids. Free fatty acids inhibit the "delayed rectifying potassium channel" in taste receptor cells. Fatty acids exhibiting these properties are those having at least two double bonds, at least one of which is in the cis confirmation, i.e., cis-polyunsaturated fatty acids. The time for taste receptor cells to

return to the "resting" state is longer following fatty acid stimulation than for other taste stimuli, thus increasing the taste receptor cell sensitivity to other stimuli, i.e., sugar or other sweet stimuli.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

SUMM To help with the growing problem of **obesity**, low-calorie food substitutes have recently been developed. Approximately 38% of the total calories consumed by most Americans come from fats. . .

SUMM A characteristic of human **obesity** and the "binge-eating" syndrome is compulsive overeating of sweet and high-fat foods. **Obese** women will often preferentially choose less sweet food that is higher in fat. Human psychophysical studies have demonstrated a correlation. . . of sweet/fat mixtures, suggesting that fat may influence the sensation of sweetness. See Drewnowski et al., "Food Preferences in Human **Obesity**: Carbohydrates Versus Fats," Appetite, Vol. 18, pp. 207-221 (1992).

DETD . . . pathway or the cyclooxygenase pathway. Arachidonic acid inhibited the delayed rectifier K+ channel in TRCs treated with the lipoxygenase inhibitor **nordihydroguaiaretic acid** (**NDGA**; 10 μM) and the cyclooxygenase inhibitor indomethacin (200 μM; n=5 cells). Furthermore, FFA-induced inhibition of the delayed rectifier K+ channel. . .

L9 ANSWER 15 OF 24 USPATFULL

Full Text

ACCESSION NUMBER: 2002:31986 USPATFULL

TITLE: Compositions and methods for improving vascular health

INVENTOR(S): Schmitz, Harold H., Branchburg, NJ, UNITED STATES
Chevaux, Kati A., Seattle, WA, UNITED STATES
Dombroski, Amy, Stanhope, NJ, UNITED STATES
Jerome, Ralph, Blairstown, NJ, UNITED STATES

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 2002018807	A1	20020214
APPLICATION INFO.:	US 2001-829782	A1	20010410 (9)

	NUMBER	DATE
PRIORITY INFORMATION:	US 2000-197135P	20000414 (60)

DOCUMENT TYPE: Utility

FILE SEGMENT: APPLICATION

LEGAL REPRESENTATIVE: Clifford Chance Rogers & Wells LLP, 200 Park Avenue, New York, NY, 10166-0153

NUMBER OF CLAIMS: 43

EXEMPLARY CLAIM: 1

NUMBER OF DRAWINGS: 8 Drawing Page(s)

LINE COUNT: 1579

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

AB This invention relates to compositions containing polyphenols, for example, cocoa polyphenols such as procyanidins, in combination with at least one cholesterol lowering agent, and methods for improving vascular health including treating and preventing atherosclerosis and cardiovascular disease.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

DETD . . . postmenoposal females w/myocardial post-ischaemic damage, surgically or chemically induced estrogen deficient females, the aged, those with hyperglycemia, diabetes, hypertension, and **obesity**, and cigarette smokers are all susceptible individuals in need of the treatment described herein. Other populations of mammals that are. . .

DETD . . . PCT/US97/05693 published as WO97/36497. Lipoxygenase Type I-B from soybean, linoleic acid (approx. 99%), Tween 20 (Polyoxyethylene-sorbitan monolaurate), and control phenols (**Nordihydroguaiaretic acid** (**NDGA**), (+)-catechin and (-)-epicatechin) were obtained from Sigma Chemical.

DETD [0072] **NDGA** is an established inhibitor of soybean and several mammalian lipoxygenases (Kemal et al, Biochemistry 26:7064-7072, 1987). It is commercially used as an antioxidant in fats and oils. **NDGA** serves as a positive control since the IC50 value of 2×10-6 M was not reached by other test phenols. (+)-catechin. . .

=> d ibib abs kwic 16-20

L9 ANSWER 16 OF 24 USPATFULL
Full Text
ACCESSION NUMBER: 2002:55057 USPATFULL
TITLE: Formulations comprising dissolved paroxetine
INVENTOR(S): Al-Ghazawi, Ahmad Khalaf Al-Deeb, Waltham, UNITED KINGDOM
Leonard, Graham Stanley, St Albans, UNITED KINGDOM
PATENT ASSIGNEE(S): SmithKline Beecham plc (non-U.S. corporation)

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 2002032220	A1	20020314
APPLICATION INFO.:	US 2001-925354	A1	20010809 (9)

RELATED APPLN. INFO.: Continuation of Ser. No. US 2000-554861, filed on 29 Jun 2000, ABANDONED

	NUMBER	DATE
PRIORITY INFORMATION:	GB 1997-24544	19971121

DOCUMENT TYPE: Utility
FILE SEGMENT: APPLICATION
LEGAL REPRESENTATIVE: GLAXOSMITHKLINE, Corporate Intellectual Property - UW2220, P.O. Box 1539, King of Prussia, PA, 19406-0939
NUMBER OF CLAIMS: 19
EXEMPLARY CLAIM: 1
LINE COUNT: 720
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB Pharmaceutical formulations of paroxetine are provided in which the paroxetine is in solution in a solid, semi-solid or liquid carrier. The solutions are used to fill capsules, or self-supporting solid solutions are shaped into solid dosage forms such as tablets or pellets. Also disclosed are novel liquid formulations in which a solubilising agent is used to solubilise paroxetine in oils and/or lipids, and methods of avoiding other paroxetine forms converting to the hemihydrate, by use of anhydrous or hydrophobic carriers or excipients.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.
SUMM . . . acid, ascorbic palmitate, thiodipropionic acid, bis hydroxy toluene (BHT), bis hydroxy anisole (BHA), gallic acid, propyl/octyl/dodecyl gallate, benzyl alcohol and **nordihydroguaiaretic acid** with or without the addition of pH modifiers and chelating agents such as citric acid and EDTA.
SUMM . . . acid, ascorbyl palmitate, thiodipropionic acid, bis hydroxy toluene (BHT), bis hydroxy anisole (BHA), gallic acid, propyl/octyl/dodecyl gallate, benzyl alcohol and **nordihydroguaiaretic acid** with or without the addition of pH modifiers and chelating agents such as citric acid and EDTA.
SUMM . . . of the paroxetine formulations of this invention include treatment of: alcoholism, anxiety, depression, obsessive compulsive disorder, panic disorder, chronic pain, **obesity**, senile dementia, migraine, bulimia, anorexia, social phobia, pre-menstrual syndrome (PMS), adolescent depression trichotillomania, dysthymia, and substance abuse, referred to below. . .

L9 ANSWER 17 OF 24 USPATFULL
Full Text
ACCESSION NUMBER: 1998:143713 USPATFULL
TITLE: Process for making spreads
INVENTOR(S): Bodor, Janos, Rijswijk, Netherlands
Patrick, Matthew, Naperville, IL, United States
Wajda, Jr., Thomas, Columbia, MD, United States
Wesdorp, Leendert Hendrik, Elliott City, MD, United States
PATENT ASSIGNEE(S): Van den Bergh Foods Co., Division of Conopco, Inc., Lisle, IL, United States (U.S. corporation)

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 5837307		19981117
APPLICATION INFO.:	US 1996-622096		19960326 (8)

RELATED APPLN. INFO.: Continuation of Ser. No. US 1995-445485, filed on 22 May 1995, now patented, Pat. No. US 5554407 which is a continuation of Ser. No. US 1994-335568, filed on 7 Nov 1994, now abandoned which is a continuation of Ser. No. US 1993-84752, filed on 29 Jun 1993, now abandoned

which is a continuation of Ser. No. US 1992-822503, filed on 17 Jan 1992, now abandoned
DOCUMENT TYPE: Utility
FILE SEGMENT: Granted
PRIMARY EXAMINER: Paden, Carolyn
LEGAL REPRESENTATIVE: McGowan, Jr., Gerard J.
NUMBER OF CLAIMS: 11
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 3 Drawing Figure(s); 3 Drawing Page(s)
LINE COUNT: 699

AB A process for preparing a very low fat water-in-oil spread and a spread made by the process. The process entails a cold mixing procedure wherein an at least partially pre-gelled aqueous phase is mixed with an at least partially pre-solidified fat continuous emulsion of fat and water. Emulsions of less than 30% fat and even 20% or less fat may be prepared.

SUMM . . . the risk of cardiac and other diseases. Moreover, the reduction of overall calories ingested has been of interest to prevent **obesity**, which has been linked to diabetes, heart disease and other ailments.
DETD Antioxidants may include normal propyl gallate, the tocopherols, including Vitamin E, butylated hydroxyanisole (BHA), butylated hydroxytoluene (BHT), **nordihydroguaiaretic acid (NDGA)**, tertiary-butylhydroquinon (TBQH) and citric acid.

L9 ANSWER 18 OF 24 USPATFULL
Full Text
ACCESSION NUMBER: 96:82485 USPATFULL
TITLE: Process for making spreads and spreads made by the process
INVENTOR(S): Bodor, Janos, Rijswijk, Netherlands
Patrick, Matthew, Naperville, IL, United States
Wajda, Jr., Thomas, Columbia, MD, United States
Wesdorp, Leendert H., Elliott City, MD, United States
PATENT ASSIGNEE(S): Van den Bergh Foods Co., Division of Conopco, Inc., Lisle, IL, United States (U.S. corporation)

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 5554407		19960910
APPLICATION INFO.:	US 1995-445485		19950522 (8)

RELATED APPLN. INFO.: Continuation of Ser. No. US 1994-335568, filed on 7 Nov 1994, now abandoned which is a continuation of Ser. No. US 1993-84752, filed on 29 Jun 1993, now abandoned which is a continuation of Ser. No. US 1992-822503, filed on 17 Jan 1992, now abandoned
DOCUMENT TYPE: Utility
FILE SEGMENT: Granted
PRIMARY EXAMINER: Paden, Carolyn
LEGAL REPRESENTATIVE: McGowan, Jr., Gerard J.
NUMBER OF CLAIMS: 10
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 3 Drawing Figure(s); 3 Drawing Page(s)
LINE COUNT: 700

AB A process for preparing a very low fat water-in-oil spread and a spread made by the process. The process entails a cold mixing procedure wherein an at least partially pre-gelled aqueous phase is mixed with an at least partially pre-solidified fat continuous emulsion of fat and water. Emulsions of less than 30% fat and even 20% or less fat may be prepared.

SUMM . . . the risk of cardiac and other diseases. Moreover, the reduction of overall calories ingested has been of interest to prevent **obesity**, which has been linked to diabetes, heart disease and other ailments.
DETD Antioxidants may include normal propyl gallate, the tocopherols, including Vitamin E, butylated hydroxyanisole (BHA), butylated hydroxytoluene (BHT), **nordihydroguaiaretic acid (NDGA)**, tertiary-butylhydroquinon (TBQH) and citric acid.

L9 ANSWER 19 OF 24 USPATFULL
Full Text
ACCESSION NUMBER: 75:45051 USPATFULL
TITLE: Aromatizing and/or antiseptic and/or oxidation inhibiting agent as well as method of producing and applying the agent
INVENTOR(S): Miler, Kazimierz B. M., Warsaw, Poland
Kozlowski, Zbigniew P., Warsaw, Poland

PATENT ASSIGNEE(S): Instytut Przemyslu Miesnego, Warsaw, Poland (non-U.S. corporation)

	NUMBER	KIND	DATE	
PATENT INFORMATION:	US 3903267		19750902	
APPLICATION INFO.:	US 1970-51668		19700701	(5)

	NUMBER	DATE
PRIORITY INFORMATION:	PL 1969-134539	19690701

DOCUMENT TYPE: Utility
FILE SEGMENT: Granted
PRIMARY EXAMINER: Waddell, Frederick E.
LEGAL REPRESENTATIVE: Waters, Schwartz & Nissen
NUMBER OF CLAIMS: 14
EXEMPLARY CLAIM: 1
LINE COUNT: 483
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

AB An agent and a method for producing an agent utilized as an additive to foodstuffs or the like for imparting thereto a smoked taste while concurrently improving their physical properties and forming an antiseptic or anti-oxidant. The method of producing the agent comprises the destructive distillation of a cellulosic or lignitic material, preferably deciduous tree wood and/or conifers or peat.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

SUMM . . . radicals or activation energy and thereby breaking definite links of the chain of autooxidation processes. Such known antioxidants are e.g. **nordihydroguaiaretic acid**, butyl hydroxyanisole, butyl hydroxytoluene, gallic acid etc. None of these agents is capable of aromatizing the products and improving their. . .

CLM What is claimed is:

. . . parts per million parts of animal feed of the agent as claimed in claim 6 to increase the rate of **weight gain** of said form animals per unit time.

L9 ANSWER 20 OF 24 USPATFULL

Full Text

ACCESSION NUMBER: 2002:39674 USPATFULL
TITLE: Pharmaceutical preparations of glutathione and methods of administration thereof
INVENTOR(S): Demopoulos, Harry B., Scarsdale, NY, United States
Seligman, Myron L., Pleasantville, NY, United States
PATENT ASSIGNEE(S): Antioxidant Pharmaceuticals Corp., Elmsford, NY, United States (U.S. corporation)

	NUMBER	KIND	DATE	
PATENT INFORMATION:	US 6350467	B1	20020226	
	WO 9829101		19980709	
APPLICATION INFO.:	US 1999-331947		19990628	(9)
	WO 1997-US23879		19971231	
			19990628	PCT 371 date

	NUMBER	DATE
PRIORITY INFORMATION:	US 1996-34101P	19961231 (60)

DOCUMENT TYPE: Utility
FILE SEGMENT: GRANTED
PRIMARY EXAMINER: Spear, James M.
LEGAL REPRESENTATIVE: Milde, Hoffberg & Macklin, LLP
NUMBER OF CLAIMS: 62
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 2 Drawing Figure(s); 2 Drawing Page(s)
LINE COUNT: 2366
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

AB A method of increasing glutathione levels in mammalian cells comprising administering an oral bolus of encapsulated pharmaceutically stabilized glutathione in a rapidly dissolving formulation to a mammal on an empty stomach. Pharmaceutical formulations including glutathione are also disclosed.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

SUMM . . . Type II. Symptoms include excessive urination, hunger and thirst with a slow steady loss of weight in the first form. **Obesity** is often associated with the second form and has been thought to be a causal factor in susceptible individuals. Blood. . .

DETD . . . high normal levels of glutathione. It is therefore believed that glutathione administration may be of benefit for the treatment of **obesity** and/or eating disorders, other addictive or compulsive disorders, including tobacco (nicotine) and opiate additions.

DETD . . . combinations of glutathione with the following drugs: cyclosporin A, thalidomide, pentoxifylline, selenium, desferroxamine, 2L-oxothiazolidine, 2L-oxothiazolidine-4-carboxylate, diethyldithiocarbamate (DDTC), BHA, nordihydroguairetic acid (**NDGA**), glucarate, EDTA, R-PIA, alpha-lipoic acid, quercetin, tannic acid, 2'-hydroxychalcone, 2-hydroxychalcone, flavones, alpha-angelicalactone, fraxetin, curcurmin, probucol, and arcanut (areca catechul).

=> d ibib abs kwic 21-24

L9 ANSWER 21 OF 24 USPATFULL

Full Text

ACCESSION NUMBER:	2001:40462 USPATFULL
TITLE:	Pharmaceutical preparations of glutathione and methods of administration thereof
INVENTOR(S):	Demopoulos, Harry B., Scarsdale, NY, United States Seligman, Myron L., Fairfield, CT, United States
PATENT ASSIGNEE(S):	Antioxidant Pharmaceuticals Corp., Elmsford, NY, United States (U.S. corporation)

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 6204248	B1	20010320
APPLICATION INFO.:	US 1999-457642		19991209 (9)
RELATED APPLN. INFO.:	Continuation of Ser. No. US 331947 Continuation of Ser. No. US 1997-2100, filed on 31 Dec 1997, now abandoned		

	NUMBER	DATE
PRIORITY INFORMATION:	US 1996-34101P	19961231 (60)

DOCUMENT TYPE:	Utility
FILE SEGMENT:	Granted
PRIMARY EXAMINER:	Reamer, James H.
LEGAL REPRESENTATIVE:	Milde, Hoffberg & Macklin, LLP
NUMBER OF CLAIMS:	14
EXEMPLARY CLAIM:	1
NUMBER OF DRAWINGS:	2 Drawing Figure(s); 2 Drawing Page(s)
LINE COUNT:	5144

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

AB A method of altering an expression of a gene product in cells or an organism, comprising orally administering glutathione in an effective amount and under such conditions to alter a redox potential in the cells. The gene expression may be sensitive to redox potential through one or more of a process of induction, transcription, translation, post-translational modification, release, and/or through a receptor mediated process. The glutathione is preferably administered as an oral bolus of encapsulated pharmaceutically stabilized glutathione in a rapidly dissolving formulation to a mammal on an empty stomach.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

SUMM . . . Type II. Symptoms include excessive urination, hunger and thirst with a slow steady loss of weight in the first form. **Obesity** is often associated with the second form and has been thought to be a causal factor in susceptible individuals. Blood. . .

DETD . . . high normal levels of glutathione. It is therefore believed that glutathione administration may be of benefit for the treatment of **obesity** and/or eating disorders, other addictive or compulsive disorders, including tobacco (nicotine) and opiate additions.

DETD . . . combinations of glutathione with the following drugs: cyclosporin A, thalidomide, pentoxifylline, selenium, desferroxamine, 2L-oxothiazolidine, 2L-oxothiazolidine-4-carboxylate, diethyldithiocarbamate (DDTC), BHA, nordihydroguairetic acid (**NDGA**), glucarate, EDTA, R-PIA, alpha-lipoic acid, quercetin, tannic acid, 2'-hydroxychalcone, 2-hydroxychalcone, flavones, alpha-angelicalactone, fraxetin, curcurmin, probucol, and arcanut (areca catechul).

L9 ANSWER 22 OF 24 USPATFULL
Full Text
ACCESSION NUMBER: 2000:167548 USPATFULL
TITLE: Pharmaceutical preparations of glutathione and methods of administration thereof
INVENTOR(S): Demopoulos, Harry B., Scarsdale, NY, United States
Seligman, Myron L., Pleasantville, NY, United States
PATENT ASSIGNEE(S): Antioxidant Pharmaceuticals Corporation, Elmsford, NY, United States (U.S. corporation)

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 6159500		20001212
APPLICATION INFO.:	US 1997-2100		19971231 (9)

DOCUMENT TYPE: Utility
FILE SEGMENT: Granted
PRIMARY EXAMINER: Spear, James M.
LEGAL REPRESENTATIVE: Milde, Hoffberg & Macklin, LLP
NUMBER OF CLAIMS: 59
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 2 Drawing Figure(s); 2 Drawing Page(s)
LINE COUNT: 2389
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB A method for the administration of glutathione orally comprising the administration of a bolus of glutathione which is pharmaceutically stabilized and encapsulated. The glutathione is administered on an empty stomach. The preferred stabilizer is ascorbic acid.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.
SUMM . . . Type II. Symptoms include excessive urination, hunger and thirst with a slow steady loss of weight in the first form. **Obesity** is often associated with the second form and has been thought to be a causal factor in susceptible individuals. Blood. . .
DETD . . . high normal levels of glutathione. It is therefore believed that glutathione administration may be of benefit for the treatment of **obesity** and/or eating disorders, other addictive or compulsive disorders, including tobacco (nicotine) and opiate additions.
DETD . . . combinations of glutathione with the following drugs: cyclosporin A, thalidomide, pentoxifylline, selenium, desferroxamine, 2L-oxothiazolidine, 2L-oxothiazolidine-4-carboxylate, diethyldithiocarbamate (DDTC), BHA, nordihydroguairetic acid (**NDGA**), glucarate, EDTA, R-PIA, alpha-lipoic acid, quercetin, tannic acid, 2'-hydroxychalcone, 2-hydroxychalcone, flavones, alpha-angelicalactone, fraxetin, curcurmin, probucol, and arcanut (areca catechul).

L9 ANSWER 23 OF 24 USPATFULL
Full Text
ACCESSION NUMBER: 1999:146636 USPATFULL
TITLE: Use of 2,4-distributed phenol derivatives as 5-lipoxygenase inhibitors
INVENTOR(S): Bonal de Falgas, Joaquin, Barcelona, Spain
Lopez Belmonte, Lorenzo, Cercedilla, Spain
Vila Navarro, Luis, Barcelona, Spain
Molins Pujol, Antonio Maria, Manresa, Spain
Lacoma Novales, Luis, Barcelona, Spain
PATENT ASSIGNEE(S): Bobel 246 S.L., Barcelona, Spain (non-U.S. corporation)

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 5985937		19991116
APPLICATION INFO.:	US 1997-997635		19971223 (8)
RELATED APPLN. INFO.:	Continuation of Ser. No. US 535125		

	NUMBER	DATE
PRIORITY INFORMATION:	ES 1994-217	19940208

DOCUMENT TYPE: Utility
FILE SEGMENT: Granted
PRIMARY EXAMINER: Jarvis, William R. A.
LEGAL REPRESENTATIVE: Striker, Michael J.
NUMBER OF CLAIMS: 17
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 4 Drawing Figure(s); 4 Drawing Page(s)
LINE COUNT: 867
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

AB The method of treating a leukotriene-mediated disease which is treatable by inhibition of 5-lipoxygenase includes administering a therapeutically effective amount of 2,4,6-triiodophenol, or a pharmaceutically acceptable salt or solvate thereof, for 5-lipoxygenase inhibition, together with an adequate amount of pharmaceutically acceptable excipients, diluents or carriers to the patient suffering from the disease. The diseases treated advantageously include, among others, herpes and gastrointestinal, respiratory, skin and/or ocular inflammatory diseases.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

SUMM . . . of such a product, what can be due to some of the following reasons: Some products, such as nordihydroguayaretic acid (**NDGA**), are very active but not enough specific (**NDGA** is a potent 5-LO inhibitor, but it is also inhibitor of all dioxygenases, and therefore cannot be used therapeutically). Other. . .

DETD . . . there was a clear and generalized disappearance of the symptoms. The case of genital herpes which resulted "regular" was an **obese** and diabetic woman, and although there was an improvement there was also reincidency. Independently an test for antiviral activity in. . .

L9 ANSWER 24 OF 24 USPATFULL

Full Text

ACCESSION NUMBER: 96:118608 USPATFULL

TITLE: Method of ameliorating cellulite by disrupting the barrier function of the stratum corneum

INVENTOR(S): Smith, Walter P., New Canaan, CT, United States

PATENT ASSIGNEE(S): Mary Kay Inc., Dallas, TX, United States (U.S. corporation)

	NUMBER	KIND	DATE	
PATENT INFORMATION:	US 5587396		19961224	
APPLICATION INFO.:	US 1994-296513		19940826	(8)

DOCUMENT TYPE: Utility

FILE SEGMENT: Granted

PRIMARY EXAMINER: Geist, Gary

ASSISTANT EXAMINER: Carr, Deborah D.

LEGAL REPRESENTATIVE: Finnegan, Henderson, Farabow, Garrett & Dunner, L.L.P.

NUMBER OF CLAIMS: 15

EXEMPLARY CLAIM: 1

LINE COUNT: 1403

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

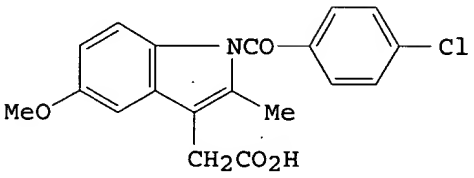
AB New topically applied treatments for cellulite are shown by comparative data to effect structural improvements in cellulite-afflicted thigh area tissues including skin-thickening, thigh-firming and thigh-reduction. The disclosed treatments disrupt the skin's water barrier and elevate trans-epidermal water loss (TEWL) for extended periods of weeks or months and include methods of mechanical or solvent action, for example, tape stripping, or acetone washes. Preferred treatments use creams with active ingredients such as lactic acid to elevate TEWL, a retinoid, preferably vitamin A palmitate to disrupt barrier rebuilding and prolong elevation of TEWL levels, and a cerebroside to inhibit lipid synthesis and intensify the TEWL elevation. Diuretics, for immediate esthetic improvements, anti-irritants and anti-oxidants for irritation control are optional ingredients.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

SUMM . . . back, torso and midriff and is not usually significant on the face, neck, hands and feet. While often associated with **obesity**, cellulite may also manifest itself in the skin of individuals of normal or near-normal weight. It is more prevalent on. . .

DETD . . . Examples of suitable anti-irritants are kola extract, green tea, aloe, and the like and examples of suitable anti-oxidants are BHT, **NDGA**, vitamins E and C, and propyl gallate.

ACCESSION NUMBER: 1981:95811 HCAPLUS
DOCUMENT NUMBER: 94:95811
TITLE: Differentiation of ob 17 preadipocytes to adipocytes. Triggering effects of clofenapate and indomethacin
AUTHOR(S): Verrando, P.; Negrel, R.; Grimaldi, P.; Murphy, M.; Ailhaud, G.
CORPORATE SOURCE: Cent. Biochim., Univ. Nice, Nice, 06034, Fr.
SOURCE: Biochimica et Biophysica Acta (1981), 663(1), 255-65
CODEN: BBACAQ; ISSN: 0006-3002
DOCUMENT TYPE: Journal
LANGUAGE: English
GI



I

AB Conversion of ob 17 preadipocytes to mature **adipose** cells was accelerated by addn. of clofenapate [21340-68-1] or of indomethacin (I) [53-86-1], in either the absence or presence of insulin. Triacylglycerol-pathway enzymes were stimulated, endogenous fatty acid synthesis was increased. This increase was a function of drug concn. and exposure time. In contrast to indomethacin, the continuous presence of clofenapate after the cells reached confluence was required to observe the effect on **adipose** conversion. Growth of ob 17 fibroblasts in the presence of 5-bromo-2'-deoxyuridine normally prevents their differentiation to **adipose** cells. Addn. of either clofenapate or indomethacin to these cells at confluence overrode this block. The effects of hypolipidemic drugs such as clofenapate obsd. on a long-term basis in vitro are consistent with the results of studies on **adipose** tissue in vivo

End of Result Set

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L1: Entry 3 of 3 File: DWPI

DERWENT-ACC-NO: 1966-20834F
DERWENT-WEEK: 196800

TITLE: Nor-dihydroguaiaretic acid compositions

PATENT-ASSIGNEE: LAB PERRIER (LPER)

PRIORITY-DATA: 1964FR-0981050 (July 8, 1964)

PATENT-FAMILY:

PUB-NO	PUB-DATE	LANGUAGE	PAGES	MAIN-IPC
FR 3866 M			000	

ABSTRACTED-PUB-NO: FR 3866M
BASIC-ABSTRACT:

Detoxicant medicament compositions contng. nor-dihydroguaiaretic acid (beta, gamma-diMe-alpha, -bis-(3,4-diHO-phenyl)-butane)

I is well known as an industrial anti-oxidant for oils and fats. The invention deals with new pharmaceutical uses as an oral detoxicant, mixed, if required, with reduced glutathione and/or vitamin B6. The principal indications are: endogenous and exogenous intoxications; hepatitis, hepatic insufficiency, pre-cirrhosis and cirrhosis, prevention of nervous and hepatic incidents from alcoholism; degeneration of conjunctive tissue; stimulation of metabolism; excessive fatiguableness.

ABSTRACTED-PUB-NO: FR 3866M
EQUIVALENT-ABSTRACTS:

DERWENT-CLASS: B00
CPI-CODES: B10-E02; B12-C06; B12-G02; B12-J01; B12-J05; B12-M06;